Short Communication: Effects of a Monensin Premix on Milk Fatty Acid Content During Subacute Ruminal Acidosis in Dairy Cows

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ABSTRACT

The effects of a monensin premix on milk fatty acid content during grain-induced subacute ruminal acidosis (SARA) in Holstein cows receiving a total mixed ration was investigated. Six multiparous, rumen-fistulated Holstein cows were used in a two-treatment, two-period crossover design with 6-wk periods. Experimental treatments were either a monensin premix or a placebo premix. At the beginning of wk 4, SARA was induced in experimental cows for a 10-d period using a grain challenge model. The administration of a monensin premix elevated milk fat proportion of total short-chain saturated fatty acids (sum of C₄ to C₁₅). Milk fat proportions of conjugated linoleic acid isomers were unaffected. Linolenic acid (C_{18:3n3}) proportion in milk fat of monensin-treated cows were lower when compared with placebo-treated cows during the SARA period. Results from this study indicate that dietary supplementation with monensin during SARA had little effect on milk fatty acid content.

(**Key words:** dairy cow, monensin, ruminal acidosis, milk fatty acids)

Abbreviation key: FA = fatty acids, PUFA = polyunsaturated fatty acids, SARA = subacute ruminal acidosis, SCSFA = short-chain saturated fatty acids

Dairy cows that are fed lactational diets rich in readily fermentable carbohydrates commonly experience subacute ruminal acidosis (SARA), a digestive disorder that is characterized by low ruminal pH (usually <5.6) (Owens et al., 1998). In beef feedlot diets,

monensin, an ionophore, is routinely added because it attenuates ruminal acidosis (Cooper and Klopfenstein, 1996). In transition dairy cows, Green et al. (1999) reported that monensin increased ruminal pH. Monensin exerts its effects primarily by altering ruminal fermentation pattern, including a decrease in acetate and butyrate production, and an increase in propionate production (Schelling, 1984). In addition, limited studies have indicated that monensin interferes with ruminal lipid metabolism by inhibiting lipolysis and biohydrogenation of dietary unsaturated fatty acids (FA) (Fellner et al., 1997), thereby increasing the content of unsaturated FA content in ruminal bacteria (Wakita et al., 1989) and the flow of unsaturated FA to post-ruminal sites (Kobayashi et al., 1992). Due to decreased ruminal biohydrogenation of dietary unsaturated FA with monensin supplementation, Sauer et al. (1998) reported a decrease in saturated FA concentration and an increase in C_{18:1} content of milk fat in dairy cows fed monensin. Additionally, high concentrate diets have also been reported to depress lipolysis and biohydrogenation of dietary unsaturated FA by ruminal contents in vitro (Van Nevel and Demeyer, 1996). The objective of the present experiment was to determine the effects of monensin on milk FA composition in dairy cows that were subjected to SARA. As both monensin and SARA have been reported to inhibit ruminal lipolysis and biohydrogenation, and monensin supplementation has also been reported to attenuate SARA, we anticipated that dietary supplementation with monensin in dairy cows subjected to SARA would increase lipolysis and biohydrogenation, thereby increasing milk concentrations of short-chain saturated FA (SCSFA) and decreasing milk concentrations of polyunsaturated FA (PUFA).

Six multiparous Holstein dairy cows (668 ± 46 kg BW; 150 ± 53 DIM) were used in this study. Animals were cared for and handled in accordance with the

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Canadian Council on Animal Care regulations, and the University of Guelph Animal Care Committee approved their use for this experiment. The experimental procedures have already been described (see Mutsvangwa et al., 2002). Briefly, the experiment was run as a two-treatment, two-period crossover design with 6-wk periods. Experimental treatments were either a monensin premix (RUMENSIN, Elanco, Division Eli Lilly Canada Inc., Guelph, ON) or a control premix. Diet composition and chemical analysis and feeding management were conducted as described by Mutsvangwa et al. (2002). Between d 22 and 32 of each experimental period, SARA was induced by feeding additional grain as described by Mutsvangwa et al. (2002).

During the 10-d SARA period, milk samples were collected without preservative daily from morning and afternoon milkings and stored at -20°C . At the end of each period, frozen milk samples were thawed in a 38°C water bath and mixed according to AOAC (1990) method 925.21. Milk samples were pooled daily based on morning and afternoon milk yields for each cow. For milk FA analysis, total milk fat was extracted with chloroform/methanol/water in the ratio of 1:1:0.9 as described by Bligh and Dyer (1959). The total lipids were methylated using NaOCH_3 as catalyst (Kramer et al., 1997; Chouinard et al., 1999) and analyzed directly by gas chromatography. A Hewlett-Packard, model 5890 Series II GC (Palo Alto, CA) was used, equipped with a flame-ionization detector, a 100-m CP Sil 88 column (Chrompack Inc., Middelburg, The Netherlands), an autosampler and a computer software system (Hewlett-Packard ChemStation A.07), and operated in a split-less mode (0.3 s), with inlet and detector temperatures set at 250°C , and a temperature program from 45 to 215°C [45°C (hold 4 min), rate of $13^{\circ}\text{C}/\text{min}$ to 175°C (hold for 27 min), rate of 4°C to 215°C (hold for 35 min)]. The FA methyl esters and the methyl esters of conjugated linoleic acids were identified by comparison with standards from Nu Chek Prep (Elysian, MN), and complementary techniques (Kramer et al., 2001).

Analysis of variance was conducted using the SAS general linear models procedure (SAS, 1990) using the following general model:

$$Y_{ijk} = \mu + \alpha_i + \beta_j + \pi_k + \varepsilon_{ijk},$$

where Y_{ijk} = the dependent variable, μ = overall mean, α_i = effect of cow ($i = 1 \dots 6$), β_j = effect of period ($j = 1, 2$), π_k = effect of treatment ($k = 1, 2$), and ε_{ijk} = random residual error. Effects were considered significant at a probability $P < 0.05$, unless otherwise indicated.

Results on DMI and milk production parameters have already been published (see Mutsvangwa et al., 2002). Briefly, supplementation with monensin premix increased DMI (21.3 vs. 19.8 kg/d; $P = 0.03$) and milk yield (31.0 vs. 27.0 kg/d; $P = 0.04$). Milk fat content (3.18 vs. 3.38%) was lower ($P = 0.03$) in monensin-treated cows compared with control cows; however, milk fat yield (0.98 vs. 0.88 kg/d) was unaffected ($P = 0.12$) by the administration of monensin (Mutsvangwa et al., 2002). The dietary addition of monensin increased ($P = 0.02$) milk fat proportion of total SCSFA (sum of C_4 to C_{15}) compared with control cows (Table 1). We had anticipated that dietary supplementation with monensin in dairy cows subjected to SARA would attenuate the condition (i.e., SARA), thereby increasing ruminal lipolysis and biohydrogenation. Such a response would be expected to increase milk fat proportions of saturated FA. In the present study, only the milk fat proportion of SCSFA was increased by feeding monensin; however, these SCSFA are mainly derived from de novo synthesis in the mammary gland from acetate and β -hydroxybutyrate (Kennelly and Glimm, 1998). In addition, all indicators of SARA that we measured, including mean ruminal pH, minimum ruminal pH and duration of time when ruminal pH < 6 and < 5.6 , were not different between control and monensin-treated cows (see Mutsvangwa et al., 2002), suggesting that monensin was ineffective in attenuating SARA. Therefore, the mechanism for increased SCSFA proportion in milk fat of monensin-treated cows is unclear. It is plausible that there was some unknown interaction between monensin and SARA such that monensin provided a prophylactic effect that allowed a more rapid adaptation to grain feeding (Mutsvangwa et al., 2002), thereby minimizing the depression in ruminal lipolysis and biohydrogenation of dietary unsaturated FA.

The administration of monensin had no effect on total milk fat proportions of $C_{18:1}$ *trans* ($P = 0.80$) and $C_{18:1}$ *cis* ($P = 0.42$) isomers (Table 1). Total milk fat proportions of conjugated isomers of $C_{18:2}$ (CLA; $P = 0.39$), and $C_{18:2}$ *cis*-9, *trans*-11 ($P = 0.22$) and $C_{18:2}$ *trans*-10, *cis*-12 ($P = 0.64$) were not affected by the dietary addition of monensin (Table 1). In previous research where milk fat depression has been observed in dairy cows, the CLA isomer *trans*-10, *cis*-12 has been identified as causing the milk fat depression (see Baumgard et al., 2000). In the present study, we observed a lower milk fat content in monensin-treated cows (3.18%) compared with control cows (3.38%) (see Mutsvangwa et al., 2002), but it is not clear what is responsible for this inhibition of milk fat synthesis because milk fat concentration of the CLA isomer *trans*-10, *cis*-12 was unaffected by the addition of mo-

Table 1. Milk fatty acids profile (expressed as g/100 g of milk fat) during subacute ruminal acidosis in Holstein dairy cows as affected by supplementation with rumensin premix.

Fatty acids	Control	Rumensin	SE	P Value
C _{4:0} to C _{15:0}	26.52	27.57	0.19	0.02
C _{16:0}	28.20	28.92	0.31	0.18
C _{16:1}	1.69	1.63	0.07	0.61
C _{17:0}	0.60	0.59	0.02	0.86
C _{17:1}	0.23	0.19	0.02	0.35
C _{18:0}	8.43	7.98	0.28	0.32
C _{18:1, trans}	4.06	4.14	0.19	0.80
C _{18:1, cis}	19.81	19.52	0.23	0.42
C _{18:2, conjugated (CLA)}				
Total	0.73	0.77	0.03	0.39
c9, t11	0.57	0.63	0.03	0.22
t10, c12	0.020	0.019	0.001	0.64
C _{18:2n6}	2.40	2.36	0.06	0.65
C _{18:3n3}	0.41	0.36	0.01	0.01
C _{20:5n3}	0.024	0.020	0.002	0.15
C _{22:6n3}	0.018	0.017	0.001	0.64

nensin. There is the possibility that the mechanism for milk fat depression in monensin-treated cows involved changes in precursor supply, as reflected by a lower acetate:propionate ratio in ruminal contents (see Mutsvangwa et al., 2002), rather than inhibition of de novo fat synthesis by CLA isomers. Milk fat proportion of linoleic acid (C_{18:2n6}) was not affected by the addition of monensin, but milk fat proportion of linolenic acid (C_{18:3n3}) was lower in monensin-treated cows compared with placebo-treated cows. Long-chain PUFA in milk originate from uptake of preformed FA from blood that are absorbed from the rumen (Kennelly and Glimm, 1998). Long-chain PUFA like C_{18:3n3} are subjected to ruminal biohydrogenation in the rumen (Kepler and Tove, 1967), with the major end-product being C_{18:0} (Loor et al., 2002). Results from the present study suggest that monensin supplementation decreased C_{18:3n3} supply to the mammary gland, presumably by increasing the rate of ruminal biohydrogenation of C_{18:3n3}, but unchanged milk fat proportion of C_{18:0} in monensin-treated cows does not lend support to this suggestion. In summary, the administration of a monensin premix had little effect on milk FA profiles when cows were subjected to SARA.

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